NIDCD Inside Newsletter

National Institute on Deafness and Other Communication Disorders
National Institutes of Health
www.nidcd.nih.gov

Summer 2006

Crank It *Down*

Your mother was right when she told you to turn down the volume. Too much noise can permanently damage your hearing. No matter how old or young you are, and no matter whether it's loud music coming from an MP3 player, the sudden blast of a hunting rifle, or the roar of a lawn mower, exposure to loud sounds can be harmful.

Photos Credit: photos.com

Noise-induced hearing loss (NIHL) is a serious problem. Some 30 million Americans are at risk for NIHL in the workplace, in recreational settings, and at home. In fact, it is the most common work-related disorder. Already 22 million Americans between the ages of 20 and 69 have permanently damaged their hearing by exposure to loud sounds or noise in their environment. Research also is finding an ever-increasing number of young people who are experiencing hearing loss to the same degree as that typically found in older adults.

When you think about noise, remember this: How loud? How close? How long? The blast of a firecracker experienced at close range can damage hearing permanently in an instant. Repeated exposures to noises from engines and machines such as motorcycles can erode hearing more slowly and so can long hours spent listening to loud portable music players.

If you are a construction worker, farmer, factory worker or airline employee, harmful sounds may be a regular part of your job. Harmful noises at home include those from vacuum cleaners, gas-powered lawn mowers, leaf blowers, and shop tools.

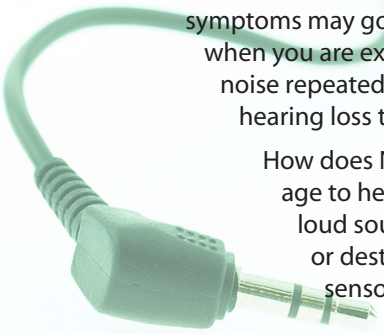
Some noisy recreational activities include target shooting and hunting, snowmobiling, go-cart riding, woodworking, and playing certain computer games. Even some children's toys have been found to produce sounds in the danger zone.

How loud is too loud? Prolonged exposure to sounds louder than 85 decibels (dB) can cause gradual hearing loss. A decibel is a unit that measures the intensity of sound. The predominant range of human hearing is represented on a scale from zero to 140 dB. A normal conversation is about 60 dB. Many personal stereo systems played at maximum volume are over 100 dB. Rock concerts and firecrackers can be 140 dB and higher.

NIHL is 100 percent preventable, but once it happens, the hearing loss can be permanent.

NIHL usually happens slowly and there is no pain. Right after exposure to noise, you may notice some "ringing" in your ears. You might have trouble hearing people talk. After several hours or even a few days, these symptoms may go away. However when you are exposed to loud noise repeatedly, you could have hearing loss that lasts forever.

How does NIHL cause damage to hearing? Exposure to loud sounds can damage or destroy the inner ear's sensory hair cells.



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IN THIS ISSUE

Once damaged, hair cells cannot grow back on their own. Scientists once believed that NIHL damages the hair cells by the pure force of the loud sound vibrations. Recent studies, however, have found that exposure to loud noise triggers the formation of free radicals-- molecules that cause damage to cells and are known to kill hair cells. Scientists supported by NIDCD have demonstrated that antioxidants, such as aspirin and vitamin E, when given as long as three days after noise exposure can protect against the damage caused by free radicals and significantly reduce hearing loss in guinea pigs. Research is needed to determine if similar results can be obtained in humans.

NIDCD-supported researchers also have learned that gene therapy may one day be used to help restore lost hearing. Gene therapy is the addition or deletion of genes, in this case those that are involved in the regrowth of hair cells. Gene therapy was found to restore hearing in animals that had been deafened by drugs that damage the inner ear. These and other efforts bring scientists closer to the development of new ways to prevent and treat hearing loss.

What can you do to prevent NIHL? All individuals should understand the hazards of noise and how to practice good hearing health in everyday life. As parents you can encourage your children to wear hearing protection in noisy environments. You also can set a good example by turning down the volume levels on all household noise sources, and by wearing hearing protection when you mow the lawn, vacuum the house, blow dry your hair, or operate power tools.

If you buy your children an MP3 player, take the time to show them how to protect their hearing from permanent damage.(Some researchers suggest that the volume should be no higher than 60 percent of the maximum and listening time should be limited to no more than one hour a day).

The good news is that you can make "hearing health" a part of your lifestyle. Know which noises are harmful and carry ear protection with you. It's up to you to prevent noise-induced hearing loss.

Photos Credit: photos.com

You Can Protect Your Hearing!

- Know which noises can cause damage.
- Wear earplugs, earmuffs, or other protective devices when involved in a loud activity.
- Teach your children to lower the volume on their portable music players and to limit listening time.
- Be alert to hazardous noise in the environment.
- Protect children who are too young to protect themselves.
- Tell family, friends, and colleagues about the hazards of noise.
- If you think you have a hearing loss, see your doctor. He or she may refer you to an otolaryngologist, or a physician who specializes in diseases of the ears, nose, and throat, or to an audiologist, who will perform a hearing test to assess the type and degree of loss.

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Innovative Surgery on the Ear Bone Relieves Rare Form of Severe Dizziness and Hearing Loss

Photo Credit: photos.com

Researchers supported by NIDCD have shown that the symptoms that accompany superior canal dehiscence, a rare hearing and balance disorder, can be treated successfully by a single operation that plugs up a thinning layer of bone in the inner ear. In what is believed to be the largest follow-up analysis of patients after their surgery for the syndrome, a team of researchers from the Johns Hopkins University Department of Otolaryngology--Head and Neck Surgery, led by Lloyd B. Minor, M.D., found that plugging the superior canal where the bone casing is thin, and then covering the plug with a bone graft, prevented symptoms from recurring.

"The surgical plugging procedure can put a stop to even severe symptoms and can lead to normal daily activities and, in some cases, to a mild-to-moderate improvement in hearing," says Dr. Minor, who, in 1995, first clinically described superior canal dehiscence and subsequently developed the surgical techniques to repair it.

Superior canal dehiscence is a rare and debilitating disorder marked by an array of disturbing symptoms, such as sudden dizziness; loss of balance; rapid, uncontrollable eye movements; falling down after a loud noise; and hearing loss. Superior canal dehiscence occurs in roughly equal numbers of men and women and is often not diagnosed until after age 40, when symptoms, such as hearing loss, appear to worsen. However, patients often recall that initial symptoms occurred much earlier in their lives.

The researchers say that the plugging procedure, which essentially compresses and closes off the canal, may help restore the hearing of patients who already have experienced some hearing loss from the syndrome. The researchers observed that improvements in hearing occurred in five of 29 patients who underwent surgery for the disorder from 1996 to 2005.

Lloyd B. Minor, M.D.

No wider than a toothpick, the canal bone rests at the top of the inner ear's three semicircular canals. Thinning due to failure of bone to develop properly and maintain strength over time makes the ear hypersensitive to sound and motion. A common complaint is autophony, where patients hear their own voice reverberating inside their head. For some patients, even a conversational level of loudness can produce symptoms strong enough to cause severe discomfort.

Although research on the condition is relatively new, Minor believes the underlying cause of the syndrome is an opening in a layer of bone that fails to develop to normal thickness during or after birth. The open canal then responds to sounds and to pressure changes from activities, such as coughing, sneezing, or straining. This abnormal activation of the canal is misinterpreted by the brain as a head movement causing patients to lose their balance and to have abnormal eye movements.

While no survey exists to indicate just how widespread this condition is, Minor says about two-thirds of those who have superior canal dehiscence never will need surgery. But for the remaining one-third who undergo surgery to repair the bone casing in the ear, the surgery in most cases fixes the disturbance in balance. In their latest findings, the researchers report that of the 19 patients with the condition who had undergone analysis of abnormal eye movements, surgery resolved each case of dizziness brought on by loud noises.

The surgical plugging procedure can put a stop to even severe symptoms and can lead to normal daily activities and, in some cases, to a mild-to-moderate improvement in hearing.

The surgery, which takes from four to six hours, is extremely delicate and requires access to the inner ear, a space the surgeons describe as no wider than the diameter of a dime. They first cut a hole above the ear and open the skull, after which they gently move aside a part of the brain so that they can reach the three paired canals. The superior canal is closest to the top of the head. The plug mixture is made up of the patient's bone and fibrous tissue taken from the area of the incision and the temporary opening the surgeons make in the skull.

"This innovative surgery offers real hope for the victims of this debilitating syndrome" says James F. Battey Jr., M.D., Ph.D. director of NIDCD. "These studies have proven that relief is possible for those individuals who have symptoms severe enough to interfere with normal activities."

The researchers note that only long-term information from post-surgical monitoring of patients for more than 10 years after surgery truly will confirm if the benefits of surgical treatments remain or decline at some point. Since the team saw its first patient with the condition in 1995, they have successfully operated on 39 individuals. The team plans to continue monitoring its patients.

The findings from this research were reported at the Combined Otolaryngological Spring Meeting held in Chicago, IL, on May 19–22, 2006.

In Drug Design, a Loose Fit May Be Best Bet

This research provides information that is fundamental to cellular signaling, a function that is essential to all cells in all systems in the body, including our sensory systems of hearing, balance, taste, and smell.

Chemical knockoffs resembling a key thyroid-related hormone are, in certain cases, more effective than the real thing at activating the target receptor, says a new study conducted in part by researchers at the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) and NIDCD. The improved performance is related to how closely coupled the chemical and receptor are, the scientists conclude, with a loose connection being more effective than a tight one. The findings are at odds with the widely held notion that the stronger the association between a hormone and its receptor, the more effective its cellular signaling. If the findings hold true for similar hormone-receptor reactions, they could help change the way that drug therapies are designed for a host of health problems, from smell and taste disorders to heart disease, asthma, migraine, and pain. The study is published in the May 12, 2006, issue of the Journal of Biological Chemistry.

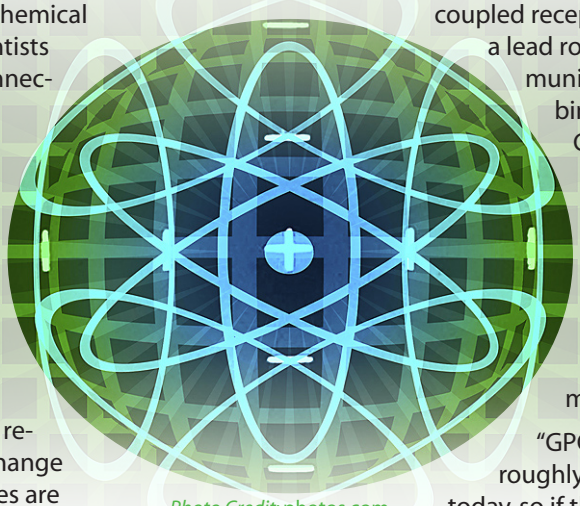


Photo Credit: photos.com

The researchers looked at thyrotropin-releasing hormone, or TRH, a hormone released in the brain that kicks off a chain of events throughout the body, including the stimulation of the thyroid gland. As with many of the body's hormones, cells recognize TRH using a receptor belonging to a mega-family of proteins known as G-protein-coupled receptors (GPCRs), which play a lead role in cell-to-cell communication. When a hormone binds to its designated GPCR on the outside of a cell, a specific G-protein is activated within the cell, initiating a cascade of biochemical events leading to the unique and appropriate cellular response to that hormone.

"GPCRs are the targets of roughly a third of medicines sold today, so if this finding for TRH holds for other GPCR targets, it could have significant implications for drug development," says Marvin C. Gershengorn, M.D., director of NIDDK's Division of Intramural Research and senior author of the paper.

Photo Credit: photos.com

"At first glance, a cellular process that affects the thyroid gland may not seem especially meaningful to the study of communication disorders," says John Northup, Ph.D., who heads the Section on Signal Transduction of NIDCD's Laboratory of Cellular Biology. "However, this research provides information that is fundamental to cellular signaling, a function that is essential to all cells in all systems in the body, including our sensory systems of hearing, balance, taste, and smell."

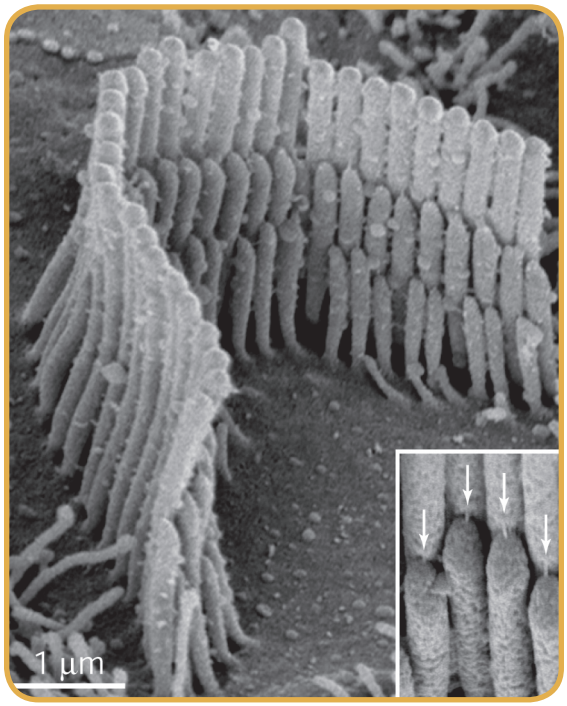
By tweaking portions of the TRH molecule, the researchers developed six slightly edited versions, while retaining most of the properties of the natural hormone. Measuring the cellular response when hormone meets receptor, they found that the lower the affinity between the two, the stronger the signal that is elicited, with certain analogs performing up to twice as effectively as TRH. As to why this would be the case, the researchers suggest that a loose connection between hormone and GPCR may allow a hormone to dock to and undock from its associated GPCR repetitively, thus activating a succession of G-proteins, and firing signal after signal. A tight connection, alternatively, may tie up a hormone with its GPCR, activating one G-protein and limiting its signaling ability.

In future studies, the scientists hope to determine whether their findings are consistent with other hormone-GPCR reactions. Other researchers taking part in the study represent the National Institute of Pharmaceutical Education and Research in Punjab, India.

Protein Tied to Usher Syndrome May Be Hearing's "Missing Link"

A protein associated with a disorder that causes deafness and blindness in people may be a key to unraveling one of the foremost mysteries of how we hear, reports a study in the June 28 issue of the Journal of Neuroscience. Scientists with NIDCD and the University of Sussex, Brighton, United Kingdom, have identified protocadherin-15 as a likely player in the moment-of-truth reaction in which sound is converted into electrical signals. (Protocadherin-15 is a protein made by a gene that causes one form of type 1 Usher syndrome, the most common cause of deaf-blindness in humans.) The findings not only will provide insight into how hearing takes place at the molecular level, but also may help us figure out why some people temporarily lose their hearing after being exposed to loud noise, only to regain it a day or two later.

"These findings offer a more precise picture of the complicated processes involved with our sense of hearing," says Elias A. Zerhouni, M.D., director of the National Institutes of Health. "With roughly 15 percent of American adults reporting some degree of hearing loss, it is increasingly vital that we continue making inroads into our understanding of these processes, helping us seek new and better treatments, and opening the doors to better hearing health for Americans."



Above: Stereocilia are arranged in three tiers atop a hair cell; Inset: Tip links connecting shorter stereocilia to their taller neighbors.

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Tapping Your Inner "Tip Link"

Researchers have long known that hair cells—small sensory cells in the inner ear— convert sound energy into electrical signals that travel to the brain through a process called mechanotransduction. However, the closer one zooms in on the structures involved, the murkier our understanding becomes. When fluid in the inner ear is set into motion by vibrations emanating from the bones of the middle ear, the rippling effect causes bristly structures atop the hair cells to bump up against an overlying membrane and to deflect. Like seats in a three-row stadium, the bristles, called stereocilia, are arranged in tiers, with each lower seat connected to a higher seat by minute, threadlike bridges, or links. As the stereocilia are deflected, pore-like channels on the surface of the stereocilia open up, allowing potassium to rush in, and generating an electrical signal. Because the "tip link"—the link that connects the tip of the shorter stereocilium to the side of the adjacent, taller stereocilium—must be present for the channel to function, scientists believe that this structure may be responsible for opening and closing the channel gate. Researchers suggest that if they can learn the makeup of the tip link, they'll be that much closer to understanding how the gate mechanism operates.

Photo Credit: photos.com

"This research identifies protocadherin-15 to be one of the proteins associated with the tip link, thus finally answering a question that has been baffling researchers for years," says James F. Battey, Jr., M.D., Ph.D., director of NIDCD. "Thanks to the collaborative effort among these researchers, we are now at the closest point we have ever been to understanding the mechanism by which the ear converts mechanical energy—or energy of motion—into a form of energy that the brain can recognize as sound."

NIDCD's Zubair M. Ahmed, Ph.D., and Thomas B. Friedman, Ph.D., together with the University of Sussex's Richard Goodyear, Ph.D., and Guy P. Richardson, Ph.D., and others used several lines of evidence to identify a protein that Drs. Goodyear and Richardson had found earlier to comprise tip links in the inner ears of young chicks. The protein is referred to as the "tip-link antigen" (TLA) because it induces the production of special antibodies, which bind to the protein at the stereocilia tips.

Using mass spectrometry, a laboratory technique that breaks down a substance into its individual components, the researchers analyzed the makeup of the TLA and found two peptide sequences that match up to key segments of the protein protocadherin-15 in humans, mice, and chickens, suggesting that the two proteins are comparable evolutionarily. Additional experiments using western blot analysis, a technique that identifies individual proteins in a substance by separating them from one another by mass and testing how they react to certain antibodies, demonstrated that the antibody that recognizes protocadherin-15 in mice also binds to the TLA.

Thanks to the collaborative effort among these researchers, we are now at the closest point we have ever been to understanding the mechanism by which the ear converts mechanical energy—or energy of motion—into a form of energy that the brain can recognize as sound.

The team also analyzed the amino acid sequences of protocadherin-15 and discovered four distinct forms — three of which are present in various developmental stages of the mouse inner ear. The researchers refer to the three alternative forms found in the inner ear as CD1, CD2, and CD3 because the sequential variations occur in the protein's "cytoplasmic domain" — a stretch of amino acids anchored inside the stereocilium. (The fourth form, referred to as SI, is likely to be secreted.) With the help of two imaging techniques that use antibodies to label a targeted protein, the team found that the distribution of protocadherin-15 along the stereocilium varies by form. The CD3 form is stationed only at the tips of the stereocilia in mature hair cells, while the CD1 form is found along the lengths of the stereocilia in mature cells but not at the tips. In contrast, the CD2 form is expressed along the lengths of stereocilia during

hair cell development, but is not present in mature hair cells.

Finally, the team found that a chemical known to break tip links—called BAPTA—had no effect on the CD1 and CD2 forms of protocadherin-15 but destroyed the CD3 form. Likewise, just as tip links are known to reappear roughly four hours after the chemical is removed, the CD3 form returned within four to 24 hours upon removal of the chemical.

Based on these findings, the researchers conclude that protocadherin-15 now can be identified as the tip-link antigen and that it is distributed in a specific way in relation to the tip-link complex. They propose that the CD3 form of protocadherin-15, located at the tip of the shorter stereocilium, may link directly or indirectly to the CD1 form on the adjacent, taller stereocilium. This scenario could help explain how tip links that are broken in real-life situations, such as from excessive exposure to loud noise, could cause temporary hearing loss until the link re-establishes itself and hearing is restored.

In future studies, the scientists plan to delve more deeply into the role that protocadherin-15 plays in the tip-link complex and whether it interacts with other components in the formation of the tip link. They also hope to determine how tip links can be stimulated to re-form, once broken.

The work was supported by NIDCD and The Wellcome Trust, London, UK. Other researchers on the project represent NIH's National Human Genome Research Institute, Bethesda, MD; University of Cambridge, UK; Brigham Young University, Provo, UT; the National Centre of Excellence in Molecular Biology, Lahore, Pakistan; and the University of Kentucky, Lexington.

Ear-Clearing Device May Replace Surgery and Drugs in Draining Middle-Ear Fluid

Each year thousands of children undergo surgery or take expensive drugs to prevent hearing loss due to a buildup of fluid in the middle ear from ear infection and other causes. Now, researchers supported by NIDCD have found that a new device, called the EarPopper, is an effective way to drain the middle ear of fluid and restore hearing.

Developed by Shlomo Silman, Ph.D., professor of hearing sciences and audiology at Brooklyn College, CUNY, and otolaryngologist Daniel Arick, M.D., the device is a new take on an old theme known as the Politzer maneuver in which a doctor injects air up one nostril of a patient while the second nostril is pinched closed and the patient swallows. During the swallow, the air is diverted into the Eustachian tube, which ventilates the middle ear. The device relieves negative air pressure and allows any accumulated fluids to drain. The EarPopper differs from the Politzer design because it provides controlled airflow and pressure in a hand-held, battery-operated device that can be used at home.

In a four-year study that included approximately 100 children, ages 4-11, researchers found that 74 percent of the children had restored hearing with the device, when used twice a day for seven weeks, compared with 24 percent improvement in the control group, which received no treatment. When the study was extended to four more weeks in patients who did not improve, the total improvement for the study group was 85 percent.

Middle-ear fluid is one of the most common reasons children visit the doctor, second only to the common cold, resulting in more than 30 million doctor visits each year and adding $4 billion in medical costs. In recent years, concerns have increased that frequent use of antibiotics for common ear conditions could raise the possibility that children could develop drug-resistant strains of bacteria in subsequent illnesses. Also, many doctors and parents want to avoid the risks of surgery which sometimes fails. Each year more than 700,000 children undergo surgery to insert tubes in their ears at an estimated cost of approximately $2000 per procedure. Tubes can fall out after several months increasing the chances of a recurrence of otitis media with effusion (OME). The researchers suggest that the EarPopper may eliminate the need for antibiotics or the placement of surgical tubes as treatments for OME , an accumulation of non-infected fluid in the middle-ear.

For more information, go to **www.earpopper.com**.

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NIDCD Highlights

Conference on Children and NIHL

There are 5.2 million children ages six to 19 who have hearing loss directly related to noise exposure.

The NIDCD is teaming up with several other organizations to sponsor the first conference on noise-induced hearing loss (NIHL) in children at work and play. Other sponsors are the Centers for Disease Control and Prevention ; the National Institute for Occupational Safety and Health ; the National Hearing Conservation Association ; the Marion Down's Hearing Center ; the Oregon Health & Science University ; and the University of Northern Colorado.

There are 5.2 million children ages six to 19 who have hearing loss directly related to noise exposure. The conference will address the following questions: How great is the risk of NIHL for our children? Are the underlying physiological mechanisms of NIHL the same in children as in adults? Is early hearing loss prevention, education and intervention successful at changing knowledge, attitudes, and behaviors of children and young adults? What responsibilities do parents, schools, employers, and public health systems have in preventing NIHL? What are the resources, programs and research agendas needed to successfully prevent NIHL in children and adolescents, both at work and at play?

Where: Embassy Suites RiverCenter Cincinnati, OH

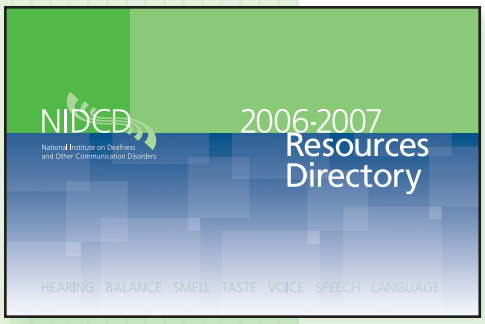
When: October 19–20, 2006

For more information and to register for the conference, go to **www.hearingconservation.org**.

Photo Credit: photos.com

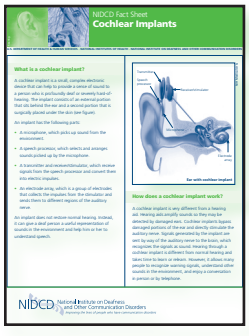
New Resources

Get Your Copy of the 2006–2007 *NIDCD Resources Directory*!



Print copies of the 2006–2007 *NIDCD Resources Directory* are now available. The directory features nearly 150 nonprofit and federal organizations committed to preventing communication disorders or to improving the lives of people who have disorders of hearing, balance, smell, taste, voice, speech, and language. Each organization is identified by up-to-date contact information as well as a short description that explains its scope and mission. To obtain a copy, contact the NIDCD Information Clearinghouse toll- free at (800) 241-1044 (voice) or (800) 241-1055 (TTY) or send an e-mail to **nidcdinfo@nidcd.nih.gov.**

Updated Cochlear Implant Fact Sheet Available



NIDCD is giving its fact sheets a makeover, with new vibrant colors, increased use of images and other graphics, and a fresh new design. Cochlear Implants is the first fact sheet to be updated. To order a copy, contact the NIDCD Clearinghouse at (800) 241-1044 or (800) 241-1055 TTY.

Meetings and Events of Interest

Look for the NIDCD exhibit at these upcoming 2006 conferences!

September 17–20	Toronto, Canada	American Academy of Otolaryngology- HNS
September 28–30	Washington, DC	American Academy of Family Physicians
October 14–18	Atlanta, GA	Society for Neuroscience
November 16–18	Miami, FL	American Speech-Language-Hearing Association

Other Meetings

Central Institute for the Deaf Fall 2006 Workshops

St. Louis, MO

September 29, 2006—*Hearing Impaired Child in the Mainstream: Evaluation, Goals, and Therapy Ideas for SLPs*

October 25–26, 2006—*SPICE-Plus: Auditory Management of Children With Cochlear Implants and Hearing Aids*

October 27, 2006—*Early Intervention for Children With Hearing Loss*
November 10, 2006—*The New Auditory-Oral Preschool*

Contact: Dianne Gushleff
Toll-free voice: (877) 444-4574, ext. 133
Voice: (314) 977-0133
Fax: (314) 977-0016
E-mail: **dgushleff@cid.edu**
Web site: **www.cid.edu**

MAAP Services for Autism and Asperger Syndrome Annual Conference

October 20–21, 2006
Adam's Mark Hotel
Indianapolis, IN
The annual conference, sponsored by MAAP Services for Autism and Asperger Syndrome and the Indiana Resource Center for Autism, MAAPing the World of ASD, will feature an all-day workshop led by keynote speaker, Tony Attwood, Ph.D., head of the Asperger's Syndrome Clinic in Queensland, Australia.

Contact:
Voice: (219) 662-1311
Web site: **www.maapservices.org**

Helen Keller National Center Training Seminars

October 23–27, 2006—*Transformation: Person-Centered Approach to Habilitation*

October 30–November 3, 2006—*The Magic of Technology:Technology Seminar*

Contact: National Training Team
Helen Keller National Center
Voice: (516) 944-8900, ext.233
TTY: (516) 944-8637
Fax: (516) 944-7302
E-mail: **ntthknc@aol.com**
Web site: **www.hknc.org**

Association for Research in Otolaryngology 30th Mid-Winter Meeting

February 10–15, 2007
The Hyatt Regency
Denver, CO

Contact:
Web site: **www.aro.org**

The National Hearing Conservation Association 32nd Annual Conference

February 15–17, 2007
Hyatt Regency
Savannah, GA

Contact:
NHCA
Voice: (303) 224-9022
Fax: (303) 770-1614
E-mail: **nhca@gwami.com**
Web site: **www.hearingconservation.org**

American Auditory Society Scientific and Technology Meeting

March 4–6, 2007
Chaparral Suites Resort
Scottsdale, AZ

Contact: Wayne J. Staab, Ph.D.
Voice: (435) 574-0062
Fax: (435) 574-0063

E-mail: **amaudsoc@aol.com**, **aas@amauditorysoc.org**
Web site: **www.amauditorysoc.org**

Stuttering Foundation of America Workshops

Five-Day Eastern Workshop: *"Diagnosis and Treatment of Children and Adolescents Who Stutter: Practical Strategies"*
June 20–24, 2007
Boston University
Boston, MA

Five-Day Western Workshop: *"Diagnosis and Treatment of Children and Adolescents Who Stutter: Practical Strategies"*
June 20–24, 2007
Portland State University
Portland, OR

Two-Week Workshop for Specialists
June 18–29, 2007
University of Iowa
Iowa City, IA

Contact:
Voice (800) 992-9392
Web site: **www.stutteringhelp.org**
Note: The Stuttering Foundation pays all tuition costs as well as room and board for these workshops. It is not too early to apply as fellowships are competitive.

Awareness Days

Hearing Aid Awareness Week

October 1–7, 2006
International Hearing Society

Contact: Alice Markey
Voice: (734) 522-7200
Fax: (734) 522-0200
E-mail: **acmarkey@ihsinfo.org**
Web site: **www.ihsinfo.org**

International Stuttering Awareness Day

October 22, 2006
Stuttering Foundation of America

Contact: Jane Fraser
Toll-free voice: (800) 992-9392
Fax: (901) 452-3931
E-mail: **info@stutteringhelp.org**
Web site: **www.stutteringhelp.org**

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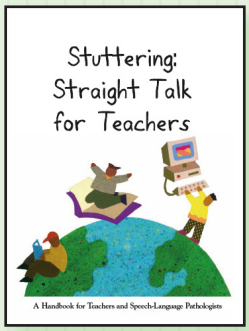
Information Exchange: News From Our Partner Organizations



Stuttering Foundation Offers Free Streaming Videos

The following videos are available free via streaming at **www.stutteringhelp.org**:

- *"Stuttering: Straight Talk for Teachers"*
- *"Stuttering: For Kids, By Kids"*
- *"Stuttering: Straight Talk for Teens"*
- *"Stuttering and Your Child: Help for Parents"*

The following Spanish-language video is available free via streaming at **www.tartamudez.org**:

- "La Tartamudez y Su Nino"

Vestibular Disorders Association (VEDA) Updates Vertigo Booklet

VEDA has updated its booklet on benign paroxysmal positional vertigo (BPPV), a condition that causes dizziness due to debris that has collected within a part of the inner ear. Topics covered include the symptoms of BPPV; the causes of BPPV, including head trauma, viral infection, and Ménière's disease; diagnostic considerations; treatment; and the impact of BPPV on one's daily life. The publication also provides a link to resource lists of physicians (both U.S. and international) who are known for treating BPPV. The cost is $4.00 for non-members or $3.00 for members. To purchase a copy, contact:

VEDA
P.O. Box 13305
Portland, OR 97213-0305
Voice: (800) 837-8428
Fax: (503) 229-8064
E-mail: **info@vestibular.org**
Web site: **www.vestibular.org**

World Federation of the Deaf Hosts 15th World Congress

July 16–22, 2007 in Madrid, Spain

Make plans to attend the World Federation of the Deaf's 15th World Congress in Madrid next summer. This World Congress will include many different programs, ranging from presentations, special interest groups, workshops and training, cultural and theater presentations, exhibits, and related events for people of all ages from all over the world.

The call for papers is posted on the World Congress Web site at **www.wfdcongress.org**.

Pre-program booklets are available from the congress's organizing committee. The deadline for early bird registration is December 31, 2006. For additional information, e-mail **wfdcongress@cnse.ed**.

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Voice: (800) 241–1044
TTY: (800) 241–1055
E-mail: nidcdinfo@nidcd.nih.gov

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